LCA Methodology • Special Issue: Air Pollution

Estimates of Real Damage from Air Pollution: Site Dependence and Simple Impact Indices for LCA*

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Abstract

In contrast to the various "potential impact" indices that have been proposed, we show that indices for real damage can be derived, based on the impact pathway methodology which involves the calculation of increased pollutant concentration in all affected regions due to an incremental emission (e.g. μg/m³ of particles, using models of atmospheric dispersion and chemistry), followed by the calculation of physical impacts (e.g. number of cases of asthma due to these particles, using a concentration-response function). The numbers are summed over all receptors of concern (population, crops, buildings, ...). We show that in a uniform world (linear dose-response function, uniform receptor density and uniform atmospheric removal rate) the conservation of matter implies a very simple formula for the total damage. The generalization to secondary pollutants is straightforward. By detailed numerical evaluations, using real data for atmospheric dispersion and geographic receptor distribution, we have demonstrated that this simple formula is an excellent representation of typical damages. Results are shown for the principal air pollutants emitted by smoke stacks of industrial installations or by road transport.

Keywords: Air pollution; damage costs; dispersion models; dose-response functions; health impacts, life cycle assessment; impact pathway analysis

1 Introduction

The goal of life cycle assessment (LCA) (HEIJUNGS et al., 1992; SETAC, 1992) is to account for all the environmental consequences, from cradle to grave, of a decision about a system, process or product (e.g. the choice between nuclear and coal-fired power plants, or the choice between plastic and glass beverage containers). While there are several variations in the implementation, the common starting point of all life cycle assessment is a careful inventory of all energy and material flows affected by a decision, including flows that are induced upstream or downstream.

LCA is the appropriate tool for collective decisions with environmental consequences. For example, a law requiring the replacement of conventional cars by electric ones would eliminate air pollution from cars at the expense of increased pollution from power plants; in addition there would be increased pollution from the manufacture of batteries and their eventual disposal. An LCA inventory answers the question whether the net change in emissions or resource use is positive or negative. However, the interpretation of the results is not straightforward except in the rare situations where all changes have the same sign. Typically a decision would improve some aspects (e.g. the emission of CO_2) while degrading others (e.g. the emission of radon caused by the mining of uranium).

To proceed, one needs some measure of the undesirability of the different environmental consequences. In the approach of Heijungs et al. (1992) this is done in two steps: the first, called classification, involves only the natural sciences, while the second, called evaluation, requires the input from the social sciences. Classification determines the contribution of the many different individual burdens (e.g. kg of CO_2, CH_4, N_2O and other greenhouse gases that are emitted) to scores for a relatively small number of categories of commonly recognized environmental effects (e.g. global warming, often quantified in terms of kg of CO_2 equivalents). The second step attempts to express the value society assigns to these effect scores. Many economists argue that the most useful measure is monetary valuation, since most decisions have to confront the reality of the market. Naturally, the evaluation step is controversial, and many in the LCA community prefer non-monetary criteria. In the present paper we present a monetary valuation as well as the non-monetary criteria (e.g. DALY = disability adjusted life years).

To evaluate the impact of a pollutant, one needs to carry out an impact pathway analysis (IPA), tracing the passage of the pollutant from the place where it is emitted to the affected receptors (population, crops, forests, buildings, etc.). The principal steps of this analysis can be grouped as follows:

1. Emission: specification of the relevant technologies and the environmental burdens they impose (e.g. kg of NO_x per MWh_e emitted by a power plant)

* A preliminary version was presented as a key note lecture at the SETAC Meeting in Bordeaux, April 14-18, 1998.

2. Dispersion: calculation of increased pollutant concentrations in all affected regions (e.g. incremental 1 μg/m³ of O_3, using models of atmospheric dispersion and chemistry for O_3 formation due to this NO_x)
3. Impact: calculation of the dose from the increased exposure and calculation of impacts (damage in physical units) from this dose using a concentration-response function (e.g. number of cases of asthma due to this increase in O_3)
4. Evaluation of these impacts (e.g. multiplication by the cost of an incident of asthma or by the number of DALY per incident of asthma).

The impacts and costs are summed over all receptors of concern.

However, most life cycle assessments have not gone far with the calculation of impacts, if they have attempted to quantify impacts at all. For example, Heijungs et al. (1992) propose general site-independent toxicity indices ("classification factors") for each pollutant. For air pollution these factors are calculated with the assumption of a uniform mixing in the earth's atmosphere. The reason for this drastic approximation lies in the emphasis on a precise account of all material flows, direct and induced. Since induced flows occur at many geographically different points under a variety of different conditions, it is not feasible to keep track of the local details of the emissions. Neglect of such site dependence is justified only for greenhouse gases and other pollutants with long residence times; it is totally unrealistic for particulate matter, NO_x, O_3 and SO_2: a ton of diesel particles emitted in the ocean is quite harmless, emitted in a big city it has severe health effects. The relation between IPA and LCA is illustrated in **Figure 1.**

In recent years the state-of-the-art of impact pathway analysis has been greatly advanced, thanks to the ExternE ("External Costs of Energy") Program of the European Commission for the analysis of fuel cycles (ExternE, 1995; 1998), carried out in conjunction with equivalent programs in the US (ORNL/RFF, 1994; Rowe et al., 1995). The purpose of the present paper is to describe the methodology used by ExternE and some of the key results that are suitable for LCA; they are concerned with air pollutants for which the dose-response functions are known. Since the results depend on the site and conditions of the pollution source (stack height, meteorological data, local and regional receptor data), we examine how the results can vary with site in order to derive general relationships. Because more than 98% of the damage costs is due to health, we document the health impact calculations in detail.

We suggest a simple way to integrate real impacts of air pollution in LCA. The key is a formula with four factors (the "uniform world model" of Eq. 9 and Eq. 14) that follows from the conservation of mass under certain simplifying assumptions. If greater accuracy is desired, one could distinguish between foreground processes and background processes, the distinction being based on the magnitude of the respective emissions. For the example of cars, the driving is a foreground process (dominant in terms of emissions), whereas the manufacture of cars is part of the background (less important). The impacts of a foreground process could be calculated with detailed site-specific dispersion models, while the "uniform world model" would be sufficient for the background processes. The "uniform world model" may even be adequate for foreground processes if suitable correction factors can be developed for the site and conditions of the pollution source.

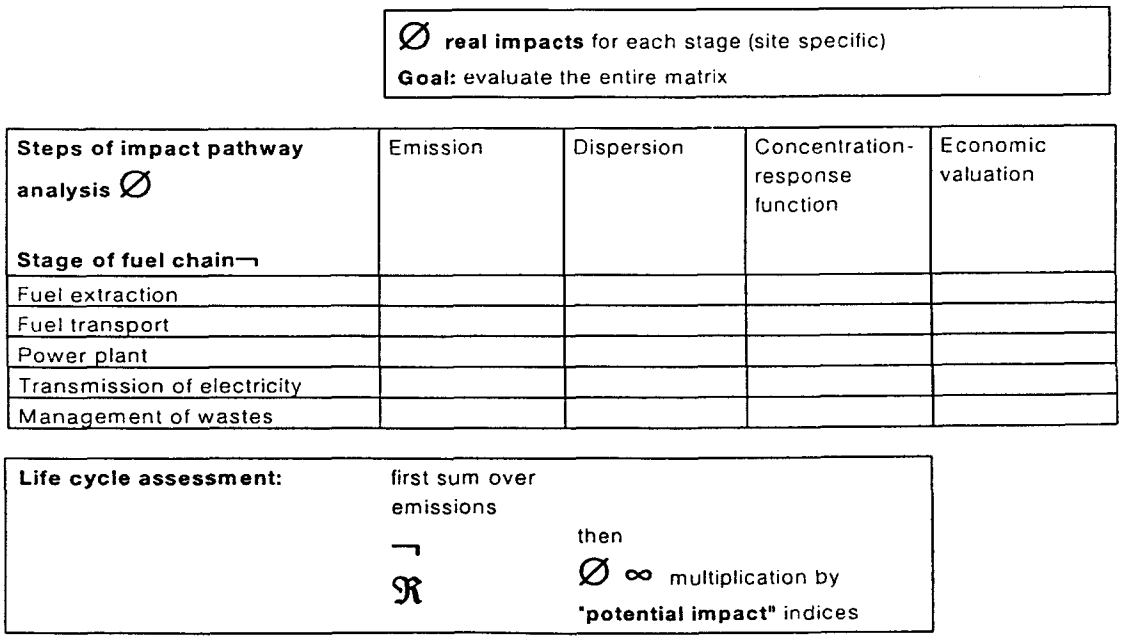

Fig. 1: Relation between impact pathway analysis and current practice of most LCA, illustrated for the example of electricity production

2 Costs and Concentration-Response Functions

2.1 Damage costs for air pollutants

Most of the impacts are incommensurate, for instance the mortality and soiling of buildings. For a comparison one needs either a multi-criteria analysis or else weighting or ranking by a common metric unit. Monetary valuation, as used by ExternE (1995; 1998), has the great advantage of yielding a single measure that is automatically consistent with the market. Note that even a non-monetary valuation cannot get around the basic problem of incommensurability; putting asthma attacks and mortality on the same scale necessarily involves a value judgment. One can reduce the incommensurability in a multi-criteria analysis by increasing the number of criteria, although at the price of a steep increase in the complexity of the analysis. In any case, subjective judgment is unavoidable.

Here we use monetary valuation. However, since some people are strongly opposed to the monetary valuation of mortality, we note that the health damage costs can be expressed in terms of equivalent physical units such as YOLL or DALY (disability adjusted life years) (MURRAY and LOPEZ, 1996). We have found that when we convert the monetary values to YOLL using the cost per YOLL, the ranking of impacts is quite close to the ranking in terms of DALY. For a detailed implementation of the DALY approach in LCA see Hofstetter (1998).

For the monetary valuation of health impacts, the single most important number is the so-called value of statistical life (an unfortunate terminology for what is really the collective willingness-to-pay for reducing the risk of premature death) for which ExternE (1998) has used

$$V = 3.1 \text{ MEuro} \approx \$\ 3.5 \text{ million}, \quad (1)$$

comparable to values in the US. A crucial question for the monetization of mortality is whether one should multiply the number of premature deaths N_{death} by V

$$\text{cost}_{Ndeaths} = N_{deaths} \times V \quad (2)$$

or whether one should base the valuation on the Years Of Life Lost (YOLL) due to a lifespan reduction ΔT

$$\text{cost}_{\Delta T} = N_{deaths} \times \Delta T \times v_{YOLL}, \quad (3)$$

where v_{YOLL} is the value of a lost year of life. This issue is particularly important for acute mortality, due to particulate matter (PM), SO_2 or O_3, which only shortens life by a brief period.

For the present paper we choose the YOLL valuation, Eq. 3, with v_{YOLL} as a fundamental unit. Its value has been derived from the value of life V as a function of the discount rate (3%) and life span reduction in the value of life studies (35 years). Unfortunately the estimation of the life span reduction introduces major uncertainties, especially for acute mortality for which ExternE (1998) assumes 9 months per premature death. For chronic mortality, the relative risk given by Pope et al. (1995) can be converted to life span reduction per exposed individual (ExternE, 1998; BRUNEKREEF, 1997; RABL, 1998), and this conversion has been assumed here.

The value of a YOLL also depends on when the loss occurs, being different for different types of mortality because of different assumptions about the respective latency as explained in ExternE (1998). We assume 0.084 MEuro per YOLL for chronic mortality and 0.155 MEuro per YOLL for acute mortality (for an explanation of these terms see Section 4.1). For the cost of a cancer we assume 1.5 MEuro, averaged by ExternE over fatal and nonfatal cancers (ExternE, 1998).

2.2 The form of the dose-response function

The form of the dose-response function is crucial for the analysis. The most important types are sketched schematically in **Figure 2** for low doses. At very high doses saturation may make the function level off in S-shaped fashion, but that regime is not relevant for typical LCA applications because the pollution levels are sufficiently low.

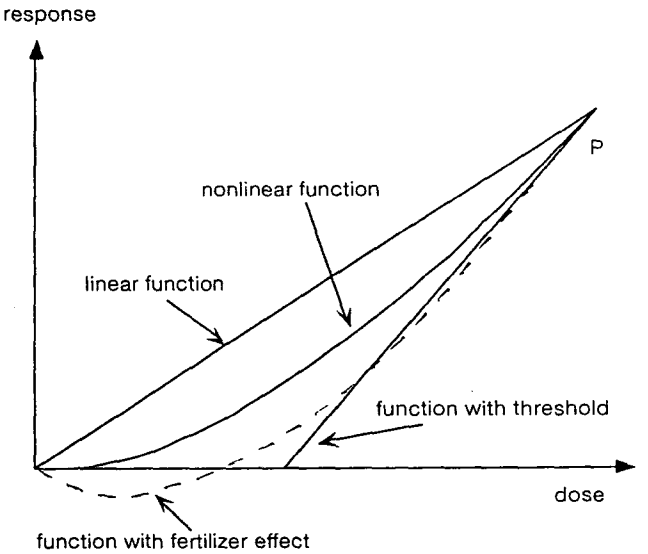

Fig. 2: Possible behavior of dose-response functions at low doses: the four functions shown have the same value at P = LOAEL (lowest observed adverse effect level)

Dose-response functions are determined from epidemiological studies or from laboratory studies. Since the latter are mostly limited to animals, the extrapolation to humans introduces large uncertainties. Another major difficulty is that one needs relatively high doses in order to obtain observable nonzero responses in a sample of realistic size; such doses are usually far in excess of the levels one is concerned

with in studies of environmental impacts. Thus there is a serious problem of how to extrapolate from the observed data towards low doses, P being the lowest observed adverse effects level (LOAEL). Figure 2 indicates several possibilities. The simplest is the linear model, i.e. a straight line from the origin through the observed data point(s). Cancer from radioactivity is an example.

Another possibility is a straight line down to some threshold, and a zero effect below that threshold. Thresholds occur when an organism has a natural repair mechanism that can prevent or counteract damage up to a certain limit. Some dose-response functions for non-cancer toxicity may be of this type.

There is even the possibility of a "fertilizer effect" at low doses, as indicated by the dashed line in Figure 2. This can be observed, for example, in the concentration-response functions for the impact of NO_x and SO_x on crops: a low dose of these pollutants can increase the crop yield, in other words the damage is negative. Such a fertilizer effect can occur with pollutants that provide trace elements needed by an organism.

If nothing is known about a threshold and if a fertilizer effect can be ruled out, the dose-response function could be anywhere between zero and the straight line extrapolation from P to the origin, for instance the curved solid line shown in Figure 2. A priori, there is no general rule about the extrapolation to low doses, except that there seem to be no pollutants with a concentration-response function above the straight line. Note that a threshold has no effect for the calculation of incremental impacts if it is below background exposure: a "hockey stick" gives the same results as a line through the origin with the same slope.

Linearity seems to be a good model for most cancers, and in the name of precaution most analysts assume linearity for all cancers. For particles, CO, SO_2, NO_x and O_3, no convincing evidence for a no-effect threshold has been found: their dose-response functions appear to be approximately linear (DOCKERY et al., 1993; DOCKERY and POPE, 1994; LIPFERT, 1994).

In view of the difficulties of determining dose-response functions for human health, it is not surprising that the available information is very limited. Apart from the ones that seem to be approximately linear, the only other information is about thresholds: either the NOAEL (no observed adverse effects level) or the LOAEL (lowest observed adverse effects level). That is typically the case for non-cancer toxicity of micropollutants such as heavy metals. However, knowing a threshold is not sufficient to quantify impacts. In theory there is no health risk for doses below a NOAEL, but that at the level of an entire population, with a wide spectrum of individual sensitivities, there may well be effects, as suggested by an analogy with the epidemiology of the classical air pollutants (particles, CO, SO_2, NO_x and O_3).

LCA, in effect, assumes linearity of all concentration-response functions: all impact indices are proportional to the emitted quantities. Linearity affords a tremendous simplification of all calculations. The absence of a true, no effect threshold also has an important implication for the dispersion calculations, because any increase in concentration anywhere entails a nonzero effect. Therefore, the geographic range of a dispersion calculation must be chosen large enough to capture most of the impact.

As for notation, many dose-response functions for air pollutants are stated directly in terms of ambient concentration rather than inhaled dose. In such cases the terms exposure-response function or concentration-response function are in use; here we employ the latter, abbreviated as CR function.

3 Atmospheric Dispersion

3.1 Dispersion models

For most air pollutants, atmospheric dispersion is significant over hundreds to thousands of km (SEINFELD and PANDIS, 1998; ZANNETTI, 1990). Both local and regional effects are important. We have therefore used a combination of local and regional dispersion models to account for all significant impacts. For modeling dispersion over the short range, we have used the ISC model (WACKTER and FOSTER, 1987).

At the regional scale, we have used two different models, the Harwell Trajectory model as employed by the ExternE Program (ExternE, 1995 and 1998), and the EMEP model of the Norwegian Meteorological Service (SANDNES, 1993; SIMPSON, 1993), the model chosen for the official allocation of acid rain budgets among the countries of Europe. The results presented here are a synthesis of calculations carried out in the ExternE Project of the European Commission (ExternE, 1995; RABL et al., 1996; ExternE, 1998; RABL et al., 1999).

We have coupled these dispersion calculations with an integration over population data, using two software packages that have been developed independently for this purpose: ECOSENSE (KREWITT et al., 1995) and PATHWAYS2.0 (CURTISS and RABL, 1996b). ECOSENSE includes the Harwell Trajectory Model; for the PATHWAYS2.0 calculations, we have used EMEP results for atmospheric dispersion. Both sets of calculations use ISC for the local dispersion. We have compared the results for total damage per kg of pollutant between these two sets of calculations and found agreement within approximately twenty percent (RABL and SPADARO, 1998).

For ozone damage due to the precursor NO_x we use a recent estimate by Rabl and Eyre (1998) based on EMEP results for the atmospheric dispersion and chemistry (SIMPSON, 1993).

3.2 Site dependence of impacts

Site dependence is particularly strong for primary pollutants (i.e. pollutants emitted by a source); this is shown by

the examples in Figure 3. For secondary pollutants (created by chemical reactions of primary pollutants) such as sulfates, nitrates and ozone, the sensitivity to local detail is lower because these pollutants are not created until some distance from the source. For nitrates and sulfates this occurs over tens to hundreds of km from the source, so that the site dependence is relatively weak; based on ECOSENSE results, we estimate that variations of sulfate or nitrate damage, per kg of SO_2 or NO_2 with a site, are around 50%. The creation of ozone is more rapid, within several km to tens of km from the source; based on EMEP data we estimate that ozone damage per kg of precursor could vary with site by about a factor of four in Europe (RABL and EYRE, 1998).

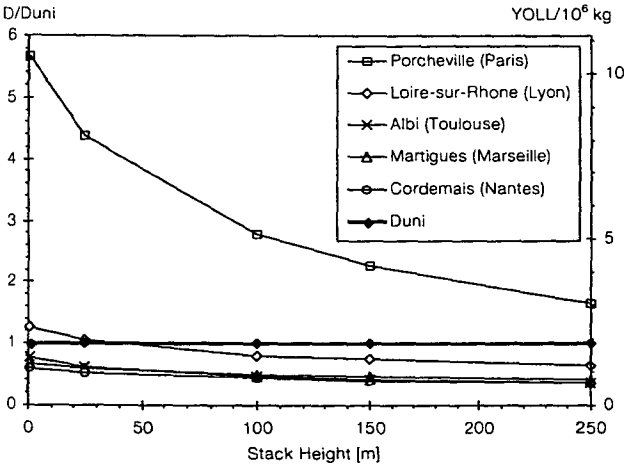

Fig. 3: An example of dependence on the site and on the height of a source for a primary pollutant with a linear CR function: damage D from SO_2 emissions, for five sites in France, in units of D_{uni} for uniform world model Eq. 9 (the nearest big city, 25 to 50 km away, is indicated in parentheses). Scale on right indicates YOLL/yr (acute mortality) from a plant with emission 10^6 kg/yr. Plume rise for typical conditions is accounted for

Figure 3 shows in fact two variations at once: with stack height, and with source location for five specific sites in France (the nearest big city, 25 to 50 km away, is indicated in parentheses). Plume rise is included for typical conditions of large combustion installations. As an example, we consider a specific impact: the increase in acute mortality (YOLL) due to an emission of Q = 10^6 kg/yr of SO_2 with the CR function of Sunyer et al. (1996). The damage is shown on two scales, as number of YOLL per year on the right hand scale, and in units of D_{uni} (to be explained below) on the left. At a stack height of 100 m, the impact for the site near Paris is about 3 times larger than D_{uni}, and for Cordemais (a relatively rural site on the Atlantic Ocean) it is about 0.4 times D_{uni}. The impact for Martigues is rather small, despite the proximity of a large city, because the prevailing wind carries the pollutants out to sea.

To explain D_{uni} (CURTISS and RABL, 1996a) let us write the damage D due to an emission Q as an integral over land area

$$D = f_{CR} \int dx \int dy\, \rho(x)\, c(x). \quad (4)$$

where
$\rho(x)$ = density of receptors at point x = (x,y),
$c(x)$ = concentration increase at x due to Q, and
f_{CR} = slope of dose-response or concentration-response (CR) function.

The slope of the CR function states the incremental number of cases (e.g. hospitalizations) per concentration increment. In the present paper, we assume that f_{CR} has a constant value independent of x.

It is instructive to relate the concentration c(x) to the depletion rate of the pollutant. There are essentially three mechanisms by which an air pollutant can disappear from the atmosphere (SEINFELD and PANDIS, 1998):

a) dry deposition (uptake at the earth's surface by soil, water or vegetation)
b) wet deposition (absorption into droplets, removed by precipitation)
c) decay or transformation (e.g. transformation of SO_2 to $(NH_4)_2SO_4$).

When evaluating the damage of a primary pollutant, this pollutant is no longer counted in the equation once it has been transformed; rather from that point on a different concentration-response function comes into play for the secondary pollutant.

The dry deposition rate is proportional to the concentration c(x) at the earth's surface, and it is customarily written in the form

$$F_{dry}(x) = v_{dry}\, c(x) \quad (5)$$

where
$F_{dry}(x)$ = deposition flux [in kg/(m²·s)], and
v_{dry} = dry deposition velocity [m/s].

Wet deposition and decay or transformation can likewise be characterized in terms of fluxes $F_{wet}(x)$ and $F_{trans}(x)$, defined as the rate at which the pollutant is removed by these mechanisms per m² (horizontal surface area) and per second. Now let us define a "depletion velocity" k(x) as a ratio of the total depletion flux

$$F(x) = F_{dry}(x) + F_{wet}(x) + F_{trans}(x) \quad (6)$$

and the surface concentration c(x) as

$$k(x) = F(x)/c(x) \quad (7)$$

the units of k are m/s. Using F(x) and k(x) we can write the damage in the form

$$D = f_{CR} \int dx \int dy\, \rho(x)\, F(x)/k(x) \quad (8)$$

Let us now consider a situation where $k(x)$ and $\rho(x)$ are independent of x. With uniform receptor density $\rho(x) = \rho_{uni}$ and uniform depletion velocity $k(x) = k_{uni}$, the integral in Eq. 8 is simply

$$D = D_{uni} = \frac{f_{CR}\,\rho_{uni}}{k_{uni}}\,Q \qquad (9)$$

because, averaged over time, the surface integral of the depletion flux equals the emission

$$Q = \int dx \int dy\, F(x) \qquad (10)$$

by conservation of matter. We shall refer to Eq. 9 (and its generalization for secondary pollutants, Eq. 14) as "uniform world model".

As an illustration we calculate D_{uni} for acute mortality due to SO_2, with f_{CR} = 5.34E-06 YOLL/(pers·yr·µg/m³) (SUNYER et al., 1996). By fitting ECOSENSE dispersion data we have found a depletion velocity k = 0.0073 m/s for SO_2. Inserting these numbers into Eq. 9 with the regional average population density ρ = 8.0E-5 person/m² (see Section 3.4) we obtain for Q = 10^6 kg/yr = 3.17E07 µg/s

$$D_{uni} = \frac{5.34 \times 10^{-6}\ \text{YOLL/(person} \cdot \text{yr} \cdot \mu\text{g/m}^3) \times 8.0 \times 10^{-5}\ \text{person/m}^2}{0.0073\ \text{m/s}}$$

$$\times\ 3.17 \times 10^{7}\ \mu\text{g/s} = 1.86\ \text{YOLL/yr} \qquad (11)$$

This is shown as the horizontal line in Figure 3. It lies right in the middle of the curves for the five sites.

Even though the assumption $k(x) = k_{uni}$ may not appear very realistic, especially near a point source, the sensitivity to deviations from uniformity is surprisingly small. The reason is that much of the total impact occurs in regions sufficiently far from the source where the pollutant is fairly well mixed vertically in the planetary boundary layer, and variations of k(x) are not too large. Emission height dependence and deviations from uniformity are most pronounced when the receptors are concentrated near the source. A source near Paris seems like a fairly extreme example because France is highly centralized, with 19% of its population in Greater Paris. Cordemais, in a semi-rural zone on the Atlantic cost, is an opposite extreme.

Thus, it is plausible that these results are fairly representative and that the simple "uniform world model" of Eq. 9 can be a useful first estimate, good to an order of magnitude. If one wants typical results for public policy, without being able to evaluate each and every site, D_{uni} seems as good a choice as any – and it has the advantage of being simple and transparent.

3.3 Secondary pollutants

The generalization of Eq. 9 for the damage D_2 due to a secondary pollutant is

$$D_{2uni} = \frac{f_{CR2}\,\rho_{uni}\,k_{1\text{-}2uni}}{k_{2uni}\,k_{1uni}}\,Q_1 \qquad (12)$$

where

Q_1 = emission rate of the primary pollutant,
f_{CR2} = CR function slope of the secondary pollutant,
k_{2uni} = depletion velocity of the secondary pollutant, and
$k_{1\text{-}2uni}$ = average of transformation velocity, defined as

$$k_{1\text{-}2}(x) = F_{1\text{-}2}(x)/c_1(x) \qquad (13)$$

with

$F_{1\text{-}2}(x)$ = creation flux of secondary pollutant per m² of horizontal surface, and
$C_1(x)$ = primary concentration increase at x due to Q_1.

We can write this in the same form as Eq. 9

$$D_{2uni} = \frac{f_{CR2}\,\rho_{uni}}{k_{2uni,\,eff}}\,Q_1 \qquad (14)$$

if we define an "effective depletion velocity"

$$k_{2uni,eff} = \frac{k_{2uni}\,k_{1uni}}{k_{1\text{-}2uni}}. \qquad (15)$$

We have determined numerical values by fits to ECOSENSE data. Table 1 shows the values of k_{uni} for different pollutants that we will use in this paper. For CO we assume 0.001 m/s based on life time estimates of 36 to 110 days given by Manahan (1994), about a factor of ten longer than for PM_{10} and SO_2.

Table 1: Values of k_{uni} for different pollutants, used in this paper for "uniform world model". Except for CO, they have been derived by nonlinear regression to dispersion calculations with ECOSENSE

a) Primary pollutants, D_{uni} of Eq. 9

Pollutant	k_{uni} [m/s]
PM_{10} (including heavy metals, dioxins)	0.0067
SO_2	0.0073
NO_2	0.0147
CO	0.001

b) Secondary pollutants, D_{2uni} of Eq. 14

Pollutant	$k_{2uni,eff}$ [m/s]
NO_2 → nitrates	0.0071
SO_2 → sulfates	0.0173

3.4 Simple indices

In the region bounded by Sicily to the South, Portugal to the West, Scotland to the North and Poland to the East, the average population density is 80 persons/km². This is about half the average EU15 population density of 158 persons/km²

per land area because it includes much water. Examining the results of detailed site-specific calculations for more than fifty installations in the EU15 countries (ExternE, 1998), we have found that the D_{uni} of Eq. 9 and Eq. 14, with k_{uni} of Table 1 and ρ_{uni} = 80 persons/km², does indeed provide representative results for typical industrial installations. In the following we therefore calculate typical values of impacts and damage costs, using k_{uni} of Table 1 and 80 persons/km².

4 Health Impacts and Costs

4.1 General remarks

A consensus has been emerging among public health experts that air pollution, even at current ambient levels, is correlated with a variety of significant health problems, especially the aggravation of respiratory diseases and increased mortality (Lipfert, 1994; Dockery and Pope, 1994; Bascom et al., 1996; Wilson and Spengler, 1996). There is less certainty about specific causes, but most recent studies have identified fine particles as a prime culprit; ozone has also been implicated directly. There may also be significant direct health impacts of SO_2. For direct impacts of NO_x, the evidence is less convincing, but this pollutant is implicated as a precursor of ozone.

Of special importance are health impacts because they account for more than 95% of the damage costs of particles, NO_x and SO_2.

To begin with, an explanation may be appropriate about the terms acute and chronic in connection with CR functions. They reflect different epidemiological approaches. The most common approach, and the easiest to implement, is to carry out a time series study of a population by identifying short term correlations (over a few days) between air pollution and a health end-point. One chooses a functional form (typically linear, logarithmic or exponential) with one adjustable parameter (more cannot be identified in practice), and determines the parameter by regression against the pollution data. Time series studies identify only short term effects and yield acute CR functions. This approach has the great advantage of being easy to implement and insensitive to the confounders (such as smoking) that plague the determination of chronic CR functions.

End-points that show up only after a longer period require observations of several populations that are exposed to different average levels of pollution. Concentration-response functions for chronic effects are notoriously difficult to establish with confidence, and there are only few studies that have determined chronic CR functions. The difference between chronic and acute CR functions is not so much in the exposure (most people are chronically exposed) as in the effects that are measured: do they show up within a few days after exposure or only after a longer period? By analogy the terms acute and chronic are also applied to CR functions for mortality, even though the attributes appear strange in that context.

In the literature, a variety of different formats have been used to describe dose-response functions. CR functions are often expressed as a % increase of an endpoint (e.g. respiratory hospital admissions) per increase in concentration. To simplify the presentation in the present paper, we adapt all CR functions to a uniform format by expressing their slope f_{CR} as number of cases per person·yr·μg/m³, i.e. per year of exposure to a given concentration, always assuming linearity. To relate exposure to emission, it is conceptually helpful to assume steady state conditions with an emission rate of 1 kg/yr and to calculate the corresponding increase in exposure. Since we have defined f_{CR} in terms of cases per year, multiplication by f_{CR} then tells us the number of cases per kg of emission.

In addition to f_{CR}, our tables show the number of cases per kg of pollutant, calculated with the uniform world model of Eq. 9, using k_{uni} of Table 1 and a population density of ρ = 80 persons/km². This is followed by the unit cost (= cost per case), by the cost per person·yr·μg/m³, and the cost per kg of pollutant.

4.2 Relation between concentration and dose

For carcinogens the customary format is in terms of dose per body weight, and a conversion is required to link it to concentration. If one makes the conservative assumption that all of the inhaled mass of a pollutant is absorbed as dose, the relation between concentration c and daily inhalation dose I is simply

$$I = c \frac{V_{inhal}}{M_{body}} \quad (16)$$

where

M_{body} = body mass (for consistency with the unit of kg we use the term mass rather than weight) and

V_{inhal} = volume of air inhaled per day.

Based on data of the EPA (1989), we take 0.42 m³/(day·kg_{body}) as the population average of the ratio inhalation volume per body mass, and relate inhalation dose I in μg/(day·kg_{body}) to concentration according to

$$I = c \times 0.42\ m^3/(day \cdot kg_{body}) \quad (17)$$

With the assumption of linearity, the dose-response functions for cancer are customarily stated as slope factor (SF). Cancer slope factors are usually estimated from human epidemiological or animal studies using mathematical models, most commonly the linearized multistage model, for estimating the largest possible linear slope (within the 95% confidence limit) at extrapolated low doses that is consistent with the data. The SF is expressed in units of mg intake per kg body mass per day. It represents the 95% upper confi-

dence limit of the probability of a carcinogenic response per daily unit intake of a chemical over 70 years. Thus the slope factor is an upper-bound estimate, and so are the risks and costs of cancer calculated in this paper. The true risk to humans, while not known, is not likely to exceed the upper bound estimate. Data on SFs can be found in the EPA's HEAST or IRIS database.

For consistency with the other CR functions in this paper we want to relate the slope factor to the number of cases per person·yr·μg/m³, i.e. per year of exposure. Conceptually it is simplest to assume steady state conditions and continuous exposure, because only total collective dose matters with a linear dose-response function, not which individual gets how much of this dose at what time. Since the slope factor is the risk for an exposure of 70 years, the risk per year is obtained by dividing by 70 years.

For example, the slope factor for inhalation of As is given by HEAST (1995) as 5.0E-2 cancers/(μg/kg$_{body}$·day), and we express it as the population averaged CR function slope f_{CR} as shown in Table 6

$$f_{CR} = SF \frac{I}{70\ yr} \frac{5.0E\text{-}2\ cancers}{\mu g/(kg_{body}\cdot day)} \frac{0.42\ m^3/(kg_{body}\cdot day)}{70\ yr}$$

$$= 3.01E\text{-}4\ cancers/(yr\cdot\mu g/m^3)\ per\ person\ exposed. \quad (18)$$

For carcinogenic heavy metals, all the slope factors of HEAST are based on inhalation doses alone; hence a conversion according to Eq. 18 is all that is needed. For dioxins, in contrast, non-inhalation pathways are very important; we include them by means of a multiplicative factor as explained in Section 4.9.

4.3 Particles

There are now numerous well-conducted studies linking particulate air pollution with a wide range of health effects, and there is no convincing evidence of a no-effects threshold (Wilson and Spengler, 1996). Particles have been associated with an increased risk of cancer and a variety of noncarcinogenic effects on the respiratory system.

There is a growing tendency to consider the associations causal, though the mechanism of action is unknown. It is not known for sure what components of respirable particulate air pollution are responsible. As for size, particles of more than 10 μm diameter are stopped in the upper respiratory ducts and appear less harmful. Between 2.5 and 10 μm, the particles penetrate more deeply into bronchi and bronchioles; particles smaller than 2.5 μm reach the alveoli of the lungs. Therefore most CR functions have been based on the PM_{10} measure; in recent years some relations have also been based on $PM_{2.5}$. We assume a ratio of $PM_{2.5}/PM_{10}$ = 0.60 based on the typical ambient concentration data in the US. We also assume that particle emissions from boilers and furnaces are almost entirely PM_{10}, as suggested by measurements of coal fired boilers (Kim et al., 1989). Our results for impacts and costs are listed in Table 2 (→ *p. 237*).

The concentration-response function for acute mortality is based on short term correlations (time scales on the order of a day) of mortality data and ambient air pollution concentrations. The certainty is relatively high (95% confidence intervals around ±50%), but only part of the mortality impact is observed. The true mortality can be significantly higher due to chronic effects that do not show up in short term correlations. Recently two important studies have been published on chronic mortality from air pollution (Dockery et al., 1993; Pope et al., 1995). These two cohort studies find clear associations of mortality with fine particles ($PM_{2.5}$) and with sulfates. Since these chronic mortality studies determine a change in age-specific mortality, one can derive implicit estimates of the YOLL (number of years of life lost) (Brunekreef, 1997; Rabl, 1998). Thus the CR for mortality function is stated in terms of YOLL in Table 2, after converting from $PM_{2.5}$ to PM_{10}.

4.4 Sulfur dioxide

The range and diversity of positive studies linking SO_2 with acute health effects is substantially greater than for NO_x, and human experimental studies are more suggestive of a causal link. The situation is not entirely clear and may be confounded by the simultaneous presence of particles. In several studies, apparent SO_2 effects disappear when particles are measured appropriately. In view of recent studies in Europe that have found an SO_2 effect, we include CR functions for acute mortality and for respiratory hospital admissions. In any case, the resulting costs, with a YOLL valuation, are relatively small. Table 3 summarizes the situation for SO_2 and NO_x, with the same format as Table 2 (→ *p. 237*).

4.5 Oxides of nitrogen

Numerous laboratory animal studies on the health effects of NO_2 have been reported. Animal studies suggest that NO_2 may have effects on host defense systems such as pulmonary clearance, immunological function and susceptibility to respiratory infection. On the other hand, the epidemiological studies are not sufficiently convincing. Even though some studies link NO_x or NO_2 with acute effects, the ExternE Program has not used these studies, because the apparent NO_x effect is arguably in reality not an effect of NO_x as such. Rather, NO_x may be a surrogate for a mixture of pollutants not measured well otherwise. NO_x, however, is implicated indirectly via nitrates and ozone.

4.6 Carbon monoxide

Carbon monoxide (CO) is certainly toxic at concentrations much higher than that found in typical urban environments;

Table 2: CR functions and costs for PM_{10}, as adapted and recommended by ExternE Program (1998). The concentration-response function slope f_{CR} has been expressed in units of cases/(person·yr·µg/m³); cost is in $Euro_{1995}$ per case. Cases/kg_{poll} and Cost/kg_{poll} according to D_{uni} of *Eq. 9* with k_{uni} of *Table 1* and ρ_{uni} = 80 persons/km²

End point for PM_{10} and reference	f_{CR} cases per (pers·yr·µg/m³)	Cases per kg_{poll}	Cost Euro/case	Cost Euro per (pers·yr·µg/m³)	Cost Euro per kg_{poll}	% of PM_{10} cost
Chronic mortality YOLL (POPE et al., 95)	4.10E-04	1.55E-04	84330	3.46E+01	1.31E+01	85.0%
CB, Adults (ABBEY et al., 95)	3.92E-05	1.48E-05	105000	4.12E+00	1.56E+00	10.1%
RAD, Adults (OSTRO, 87)	2.00E-02	7.56E-03	75	1.50E+00	5.67E-01	3.7%
Bronchodilator usage, Asthmatic adults (DUSSELDORP et al., 95)	4.56E-03	1.73E-03	37	1.69E-01	6.39E-02	0.4%
Chronic cough, Asthmatic children (DOCKERY et al., 89)	4.14E-04	1.57E-04	225	9.32E-02	3.53E-02	0.2%
CB, Asthmatic children (DOCKERY et al., 89)	3.22E-04	1.22E-04	225	7.25E-02	2.74E-02	0.2%
HA, Cerebrovascular (WORDLEY et al., 97)	5.04E-06	1.91E-07	7870	3.97E-02	1.50E-02	0.1%
Cough, Asthmatic adults (DUSSELDORP et al., 95)	4.69E-03	1.78E-03	7	3.28E-02	1.24E-02	0.1%
Congestive heart failure, Elderly 65+ (SCHWARTZ&MORRIS, 95)	2.59E-06	9.81E-07	7870	2.04E-02	7.72E-03	0.1%
Bronchodilator usage, Asthmatic children (ROEMER et al., 93)	5.43E-04	2.05E-04	37	2.01E-02	7.60E-03	< 0.1%
HA, Respiratory (DAB et al., 96)	2.07E-06	7.84E-07	7870	1.63E-02	6.17E-03	< 0.1%
LRS, Asthmatic adults (DUSSELDORP et al., 95)	1.70E-03	6.42E-04	7.5	1.27E-02	4.82E-03	< 0.1%
Cough, Asthmatic children (POPE&DOCKERY, 92)	9.34E-04	3.54E-04	7	6.54E-03	2.48E-03	< 0.1%
LRS, Asthmatic children (ROEMER et al., 93)	7.20E-04	2.73E-04	7.5	5.40E-03	2.05E-03	< 0.1%
Total PM_{10}				**4.07E+01**	**1.54E+01**	100%

HA = hospital admission; CB = chronic bronchitis; RAD = restricted activity day; YOLL = years of life lost;
LRS = lower respiratory symptoms

Note: to derive f_{CR} from the data in the references (given e.g. as % increase per receptor), we have assumed: 3.5% of the population is asthmatic, 14% of the population is over 65, and mortality rate is 990 deaths/yr per 100000; for chronic mortality f_{CR} has been obtained by integration over life tables (ExternE 1998) or the Gompertz function for age-specific mortality (RABL, 1998), assuming that it applies only to the population over 30 years of age (= cohorts in POPE et al., 1995)

Table 3: CR function slope f_{CR} and costs for SO_2 (direct and via sulfates), NO_2 (via nitrates and via O_3), and CO. Same format and assumptions as *Table 2*

End point and reference	f_{CR} cases per (pers·yr·µg/m³)	Cases per kg_{poll}	Cost Euro/case	Cost Euro per (pers·yr·µg/m³)	Cost Euro per kg_{poll}	% of total cost
SO_2 Impacts						
Acute Mortality YOLL (SUNYER et al., 96) [a]	5.34E-06	1.86E-06	155000	8.27E-01	2.88E-01	2.8%
Respiratory HA (PONCE DE LEON, 96)	2.04E-06	7.09E-07	7870	1.61E-02	5.58E-03	0.1%
SO_2 via sulfates [b]					9.95E+00	97.1%
Total SO_2					**1.02E+01**	100%
NO_x Impacts						
NO_2, via nitrates [c]					**1.45E+01**	92%
NO_2, via O_3 (RABL&EYRE, 98)					**1.15E+00**	8%
Total NO_2					**1.57E+01**	100%
CO Congestive heart failure elderly 65+, (SCHWARTZ&MORRIS, 95) [a]	7.90E-08	2.00E-07	7870	6.21E-04	**1.58E-03**	

[a] assuming YOLL = 9 months per death.

[b] obtained by multiplying total in *Table 3* by 1.67 (see Eq. 20) and by 0.67/1.73 for k's of *Table 1*

[c] obtained by multiplying total in *Table 3* by 1.0 (see Eq. 19) and by 0.67/0.71 for k's of *Table 1*

to protect human health, the ambient air quality standards of EPA and of WHO impose an upper limit of 10 mg/m³, averaged over 8 hours. However, it seems that there are harmful effects at lower concentrations, and several recent studies have proposed linear CR functions for CO. The evidence for a correlation with hospital admissions is quite strong and we have included it in Table 3. There may also be mortality impacts due to CO, but the case is less clear.

4.7 Ozone

Damage costs for ozone formation have been estimated recently by Rabl and Eyre (1998). The underlying assumptions for CR functions and cost per case are listed in Table 4. The step from cost per ppb ozone to cost per kg of precursor involves results of the EMEP model for ozone formation (SIMPSON, 1993), as explained in Rabl and Eyre (1998). The resulting costs are listed in Table 5, together with agricultural losses. Only a single European average of regional damages was derived, and we do not know how much the results would change if local ozone modeling were included.

pact calculations performed in the ExternE Program (ExternE, 1995 and 1998) which found that the damage per kg of SO_2 and per kg of NO_2 was dominated by the assumption on the toxicity of these aerosols.

In the ExternE program, the assumption has been made to apply the CR functions for particles to these aerosols per concentration of pollutant mass. In particular, nitrate aerosols are treated like PM_{10}

$$f_{CR} \text{ for nitrates} = f_{CR} \text{ for } PM_{10} \quad (19)$$

and sulfate aerosols are like $PM_{2.5}$

$$f_{CR} \text{ for sulfates} = f_{CR} \text{ for } PM_{2.5} \ (= 1.67\ f_{CR} \text{ for } PM_{10}) \quad (20)$$

the conversion factor 1.67 being used for the usual case where CR functions have been determined only with respect to PM_{10}; this conversion is based on ratios ($PM_{2.5}$ / $PM_{10} \approx 0.6$) that have been observed in ambient concentration data. The resulting damage estimates are very large, especially per kg of NO_2.

Table 4: Concentration-response function slope f_{CR} for O_3 health impacts and the corresponding costs. f_{CR} is based on 6hr concentrations (a factor 1.3 has been used to convert from 1 hr peak to 6 hr peak)

	f_{CR} cases per (person·yr·ppb) [a]	Euro per case [b]	Euro/(person·yr·ppb)	%
Acute mortality (SUNYER et al., 1996)	1.17E-05	82500 [c]	0.965	36%
HA Respiratory (PONCE DE LEON, 1996)	1.42E-05	6600	0.093	3%
MRADs (OSTRO et al., 89)	1.95E-02	62	1.209	45%
Symptoms Days (KRUPNICK et al., 90)	6.60E-02	6.3	0.416	15%
Total Euro/(yr·person·ppb)			**2.68**	100%

MRAD = Minor Restricted Activity Days, HA = Hospital Admission

[a] coefficients of Table 4.4 of ExternE (1995) and updates (ExternE 1998), multiplied by 1.3

[b] from Table 16.3 of ExternE (1995), except for acute mortality, see text below

[c] assuming YOLL = 9 months per death and 0.11 MEuro per YOLL due to O_3

Table 5: Summary of damage costs per kg of ozone precursor for average emissions in Europe, based on EMEP (SIMPSON, 1993) and on Harwell Global Ozone model (HOUGH, 1991)

Euro/kg	Health	Crops	Total
NO_2	1.150	0.350	1.50
VOC	0.734	0.196	0.93

4.8 Sulfate and nitrate aerosols

SO_2 and NO_x are transformed to sulfates in the atmosphere and thereby become a component of the suspended particulate matter. The detailed composition of these secondary aerosols depends on the local concentration of NH_3 which neutralizes sulfuric and nitric acid to form NH_4HSO_4, $(NH_4)_2SO_4$ and NH_4NO_3. There is uncertainty about the health impacts of these secondary pollutants. Their importance seems to have been recognized only in recent years, as a result of im-

In the present paper we follow these assumptions, although with a strong warning about the uncertainties. There is insufficient epidemiological evidence to identify which individual characteristics or components of particulate air pollution determine the toxicity of particles: mass, particle number, composition, acidity, oxidizing potential, solubility, ...? While the evidence from smoking suggests that primary combustion particles are harmful, the evidence about secondary aerosols is not clear.

Although there are studies that report correlations of mortality and other end points with sulfate concentrations, it is not clear to what extent the true correlation is with some other component(s) of particles. There are no CR functions for nitrates because in the past nitrates have not even been monitored as a separate component of air pollution. Furthermore, nitrates and sulfates are mixtures of components with different properties, especially different acidities. Acidity has sometimes been implicated in epidemiological stud-

ies. NH_4NO_3, in particular, is a neutral and highly soluble substance and therefore no physical irritant, while HNO_3 is a strong acid. In addition, nitrates could be harmful since they are oxidants.

4.9 Dioxins

Dioxins are among the most thoroughly studied of all of the pollutants. There are a large number of different dioxins, with different toxicity. It is convenient and common practice to express emissions and dose-response function for all dioxins (as well as the closely related furanes) in terms of TEQ = toxic equivalence 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

Several human epidemiological studies and numerous studies in experimental animals have been carried out. There can be acute as well as chronic effects. Dioxins cause changes in laboratory animals that may be associated with developmental and hormonal effects; however, the mechanism of carcinogenicity is unclear. Whether the biochemical changes may result in adverse health effects in people and at what concentrations is not very well known.

As for cancers, dioxins 2,3,7,8-TCDD was said by EPA to be "the most potent carcinogen(s) evaluated by the EPA's Carcinogen Assessment Group". In this paper, the only impacts of dioxins we quantify are cancers and we use the slope factor shown in **Table 6**. The slope factor indicates the likelihood of contracting cancer from a lifetime exposure (70 years) for a typical person (70 kg) breathing in 1 µg/m³ of a pollutant.

In calculating the dose for dioxins, one needs to take non-inhalation pathways into account, because dioxins are persistent and bioaccumulate, becoming concentrated in milk, meat and fish. Figure II-5, p. 37 of the report "Estimating exposure to dioxin-like compounds" vol.I Executive Summary (EPA, 1994a and 1994b) indicates a typical value of

$$\frac{\text{total dose}}{\text{inhalation dose}} = \frac{119}{2.2} = 54.1 \qquad (21)$$

for the ratio of total dose to inhalation dose for dioxins. The non-inhalation dose involves dispersion over large distances (e.g. by transport of cattle feed, followed by transport of meat). This has the effect of making the receptor distribution more uniform. Therefore we recommend estimating the total collective dioxin dose by calculating the damage due to inhalation in site specific manner and adding it to the "uniform world model" dose of Eq. 9, the latter multiplied by the ratio of Eq. 21 (after subtracting 1 to count only the non-inhalation dose),

$$D_{total} = D_{inhalation} + (54.1 - 1)\, fCR \frac{\rho_{uni}\, Q}{k_{uni}} \qquad (22)$$

where

D_{total} = total damage,
$D_{inhalation}$ = damage due to inhalation,
ρ_{uni} = average population density of the region,
Q = emission rate, and
k_{uni} = mean depletion velocity (= 0.0067 m/s for particles).

The term $\rho uni\ Q/k_{uni}$ is the collective inhalation dose for a uniform population density. By separating the inhalation dose as in Eq. 22 one can take into account higher population densities for sources near cities. However, if the ratio of total and inhalation dose is indeed as large as indicated by Eq. 21, the contribution of the first term in Eq. 22 is so small that site dependence can be neglected in most cases. In the present paper we therefore assume a uniform population density for the inhalation dose as well. We note that the slope factor and the ratio of total and inhalation dose are uncertain and controversial.

4.10 Other pollutants

Among the heavy metals, the following are considered carcinogenic: arsenic (As), cadmium (Cd), chrome (Cr-VI) and nickel (Ni). The corresponding toxicity data are summarized in **Table 6**. Only the inhalation dose has been taken into account because the available slope factors are for inhalation. By contrast to the classical air pollutants and carcinogens, we only have data for thresholds below which no adverse effects have been observed for non-cancer impacts of heavy metals.

Table 6: Cancer risks per kg of pollutant, calculated with D_{uni} for emission rate of 1 kg/yr, for population density of 80 persons/km²

	SF	f_{CR}	Cases	Cost		
	cancers per (µg/kg·body·day)	cancers per (pers·yr·µg/m³)	per kg_{poll}	Euro/case	Euro per (pers·yr·µg/m³)	Euro per kg_{poll}
As	5.00E-02	3.01E-04	1.14E-04	1500000	452	1.71E+02
Cd	6.10E-03	3.67E-05	1.39E-05	1500000	55	2.09E+01
Cr (VI)	4.10E-02	2.47E-04	9.35E-05	1500000	370	1.40E+02
Ni	8.40E-04	5.06E-06	1.91E-06	1500000	8	2.87E+00
Dioxin TEQ	1.00E+02	6.02E-01	1.23E+01	1500000	4.88E+07	1.85E+07

SF = slope factor
References: for As, Cd, Cr, Ni: HEAST (1992 and 1995); for dioxin: "plausible upper bound" for slope factor vol. III p.9.85 (EPA, 1994b)

4.11 Comparison with LCA toxicity index

It is interesting to compare our results with the human toxicity index that has been used in some LCA studies. In **Table 7** we show health damage costs and human toxicity index for

those pollutants where both are available. The toxicity index values in this table have been calculated during an evaluation of waste treatment technologies (EC, 1996), using the SIMAPRO software (Pré Consultants, 1995). Even though the units are totally different, the relative ranking of different pollutants should be the same, i.e. the ratio ExternE/LCA should be the same for all pollutants. It is disturbing that the rankings are very different, as shown by the large dispersion of ratios (from 0.03 to 21.8).

ferent stocks at risk at each site, as well as different background concentrations with nonlinear dose-response functions. Also, these impacts are direct effects of SO_2, rather than effects via sulfates. The combined crop and material effects account between 1% and 4% of the overall SO_2 impact, with an average contribution of 2.5% (standard deviation is one-half of the average value). In monetary terms, the average damage to crops and materials amounts to just over 0.3 Euro per kg of SO_2. This is very small compared to the public health

Table 7: Comparison of results for health impacts, between LCA and ExternE. The "human toxicity index" of LCA has been calculated using the SIMAPRO software. This software does not account for health impacts of VOC and particles. The damage costs of ExternE are typical values for Europe

Pollutant	ExternE Euro/kg_{poll}	LCA toxicity index/kg_{poll}	Ratio ExternE/LCA
PM_{10}	1.54E+01	-	-
SO_2 (direct + via sulfates)	1.02E+01	1.20E+00	**8.5**
NO_x (via nitrates)	1.45E+01	7.80E-01	**18.6**
VOC (via O_3)	7.0E-01	-	-
As	1.71E+02	4.70E+03	**0.04**
Cd	2.09E+01	5.80E+02	**0.04**
Dioxin	1.85E+07	3.30E+06	**5.6**

5 Other Impacts

Aside from health impacts, the ExternE project has evaluated damages to agricultural crops, forests, material surfaces (building facades) and ecosystems (ExternE, 1995 and 1998). Table 8 summarizes the damage costs to crops and materials for an emission source located in various urban and rural sites across Europe. The impacts are estimated by the ECOSENSE software (KREWITT et al., 1995) and include effects from SO_2 airborne exposure and wet acid deposition. Crops include the following species: barley, oats, potatoes, rye, sugar beet and wheat. For the materials category, we consider the following building surfaces: galvanized steel, limestone, mortar, natural stone, paint, rendering, sandstone and zinc. For buildings and materials, the concentration-response functions are non-linear and quite complex. A detailed description is beyond the scope of this paper; and we therefore refer the reader to ExternE (1995; 1998).

As seen in Table 8, the damage costs vary significantly between the different locations. This variability reflects the different stocks at risk at each site, as well as different background concentrations with nonlinear dose-response functions. Also, these impacts are direct effects of SO_2, rather than effects via sulfates. The combined crop and material effects account between 1% and 4% of the overall SO_2 impact, with an average contribution of 2.5% (standard deviation is one-half of the average value). In monetary terms, the average damage to crops and materials amounts to just over 0.3 Euro per kg of SO_2. This is very small compared to the public health cost of 10.2 Euro/kg of SO_2 (→ *Table 3*). Damage costs to forests appear to be even smaller than damages to crops, about 1/3 as much, although they are quite uncertain.

Only for ozone does the damage to crops appear comparable to the health damage (→ *Table 5*). This is a consequence of the ExternE assumptions about mortality impacts of ozone and particles, in particular the hypothesis that sulfates are like $PM_{2.5}$. With the chronic mortality CR function of Pope et al. (1995) this leads to a high number for SO_2. The ozone health cost is small, by contrast, because no link between ozone and chronic mortality has been established so far.

6 Results

Typical damage costs per kg of pollutant are summarized in Table 9. They correspond to the "uniform world model" with the parameters of Section 3.4, in particular a regional average (land and water) population density of 80 persons/km². For other regions the health damage costs should be

Table 8: Damage costs to crops and materials for various sites across Europe

Site	Crops		Materials	
	(% of SO_2 Cost)	Euro/kg_{SO2}	(% of SO_2 Cost)	Euro/kg_{SO2}
Albi (Fr)	0.07	0.005	1.3	0.10
Barcelona (Es)	0.20	0.020	1.5	0.15
Bordeaux (Fr)	0.31	0.036	2.5	0.30
London (UK)	0.27	0.034	4.1	0.52
Nantes (Fr)	0.05	0.004	1.0	0.09
Paris (Fr)	0.11	0.018	2.2	0.34
Piacenza (It)	0.28	0.034	2.5	0.31
Stuttgart (De)	0.24	0.032	3.0	0.39
Vienna (Au)	0.19	0.018	3.6	0.35
Average	**0.19**	**0.022**	**2.4**	**0.28**

scaled according to population density. The notes under Table 9 also give an indication of how much the damages may vary for different sites and stack conditions. For the cost per kg of $CO_{2equivalent}$ of greenhouse gases we cite the results of ExternE (EYRE et al., 1998). We also show the damage cost for particles from motor vehicles, calculated by SPADARO et al. (1998); they are much larger because of the proximity to population. In addition, the particles are more toxic by virtue of being smaller, ExternE (1998) assuming that $PM_{2.5}$ is 1.67 times more harmful than PM_{10}.

7 Discussion

We have developed a method to integrate real impacts of air pollution into LCA. The key is the "uniform world model" of Eq. 9 and Eq. 14. If greater accuracy is desired, one could distinguish between foreground processes (to be modeled in detail) and background processes (to be approximated with the "uniform world model"). The "uniform world model" may even be adequate for foreground processes if suitable correction factors can be developed for site and conditions of the pollution source.

Table 9: Typical damage costs per kg of pollutant, based on ExternE (1998). Values for large combustion installations correspond to uniform world model D_{uni} of Eq. 9 and Eq. 14 with population density 80 persons/km² (Europe). Values for $PM_{2.5}$ by cars from SPADARO et al. (1998)

Pollutant	Impact	Cost, Euro/kg_{poll}
$PM_{2.5}$ (primary) cars, Paris	mortality and morbidity	2190
$PM_{2.5}$ (primary) cars, Paris-Lyon	mortality and morbidity	159
$PM_{2.5}$ (primary) cars, rural	mortality and morbidity	21.5
PM_{10} (primary)	mortality and morbidity	15.4
SO_2 (primary)	Crops, materials	0.3
SO_2 (primary)	mortality and morbidity	0.3
SO_2 (via sulfates)	mortality and morbidity	9.95
NO_2 (primary)	mortality and morbidity	small
NO_2 (via nitrates)	mortality and morbidity	14.5
NO_2 (via O_3)	mortality and morbidity	1.15
NO_2 (via O_3)	Crops	0.35
VOC (via O_3)	Crops, mortality and morbidity	0.9
CO (primary)	morbidity	0.002
As (primary)	cancer	171
Cd (primary)	cancer	20.9
Cr (primary)	cancer	140
Ni (primary)	cancer	2.87
Dioxins, TEQ	cancer	1.85×107
CO_2	Global warming	0.029

For a given relation of source and receptors $PM_{2.5}$ is 1.67 times as toxic as PM_{10}.
Multipliers for **variation with site** (proximity of big city, local climatic conditions) and **stack conditions** (stack height h, temperature T, exhaust velocity v):
- No variation for CO_2;
- Weak variation for dioxin because non-inhalation pathways dominate: ≈ 0.7 to 1.5;
- Weak variation for secondary pollutants: ≈ 0.5 to 2.0;
- Strong variation for primary pollutants: ≈ 0.5 to 5 for site, ≈ 0.6 to 3 for stack conditions (up to 15 for ground level emissions in big city)

For PM_{10} and aerosols (nitrates and sulfates), 85% of the cost is due to mortality impacts, with the remaining 15% attributed to morbidity cases (bronchitis, asthma, etc.). If physical impact indices are preferred, one can convert to equivalent YOLL (years of life lost) using v_{YOLL} = 0.084 MEuro/YOLL. One could also use DALY (disability adjusted life years) as discussed by Hofstetter (1998). We warn that the uncertainties are large, typically a factor of 3 to 5 in either direction (RABL and SPADARO, 1998). Most of the uncertainty lies in the epidemiology.

To illustrate this method, let us consider a possible replacement of diesel passenger cars in Paris by electric cars. Obviously we do not have the space here for a complete LCA and our example is extremely simplified by considering only the dominant items: tail pipe emissions of the diesel and emissions from the power plants that provide the electricity for the electric car. Even though current base load electric power in France is entirely nuclear, we assume a gas turbine combined cycle because it appears to be the preferred option for incremental base load in France in the near fu-

Table 10: Illustration of use of the damage cost estimates of *Table 9* for LCA: comparison of diesel and electric car in a large city if electricity for the car is produced by gas turbine combined cycle. Only emissions from tail pipe and from power plant are taken into account

	Diesel car			Electric car		
	Emissions g/km [a]	Damage Euro/kg	Damage Euro/km	Emissions g/kWh [b]	Emissions g/km [c]	Damage Euro/km
Particles	0.08	2190 [d]	0.175	0	0	0.000
SO_2 (via sulfates)	0.0258	9.95	0.000	0	0	0.000
SO_2 primary		25.6 [e]	0.001			0.000
NO_x	0.511	16.0	0.008	0.7	0.175	0.003
NMVOC	0.189	0.9	0.000	0	0	0.000
CO_{2equiv}	319.2	0.029	0.009	430 [f]	107.5	0.003
Total			**0.194**			**0.006**

[a] tailpipe emissions equal to Directive 94/12/EC, March 1994
[b] emissions for new gas turbine combined cycle power plant
[c] electricity per km urban driving 0.25 kWh/km
[d] $PM_{2.5}$ in Paris
[e] primary SO_2 damage of *Table 9* corrected for Paris by multiplying by 2190/(15.4×1.67)
[f] includes upstream emissions of greenhouse gases

ture. We treat the emissions from the car as a foreground process (i.e. for the real site and conditions), while the power plant emissions are background. The results in **Table 10** suggest that the electric car entails far lower damages than the diesel car.

8 Conclusion and Future Outlook

It would be desirable to expand and improve this work, by evaluating a wider set of sites and source conditions. In particular there remains the challenge of extending this methodology to pollutants emitted to water or soil. One difficulty is site dependence, since impacts of emissions to water can be fairly site specific and impacts of emissions to soil extremely site-specific. But an even greater obstacle is the lack of concentration-response functions for non-cancer toxicity. Since impact indices have been developed in the framework of LCA, these indices could perhaps be calibrated using impacts for which concentration-response functions are known. Among future improvements of this work we emphasize the need for updating of the dose-response functions to take into account a continuing progress in epidemiology.

Acknowledgments

This work has been supported in part by the ExternE Project of the JOULE Program and by the Environment and Climate Program of the European Commission DG12, by ADEME, and by the Ministère de l'Environnement. We thank Jérôme Adnot, Roland Clift, Denis Clodic, Peter Curtiss, Mark Delucchi, William Dab, Brigitte Desaigues, Nick Eyre, Rainer Friedrich, Renaud Gicquel, Patrick Hofstetter, Mike Holland, Fintan Hurley and Wolfram Krewitt, as well as two referees for discussions and comments.

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Received: October 5th, 1998
Accepted: January 26th, 1999